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AUTHOR(S):

MUTO, KOTARO

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REFLEX SHOCK AND ITS PREDISPOSING STATE

by

KOTARO MUTO

From the 1st Surgical Division, Mie University Medical School
(Director: Prof. Dr. TOSHISUKE YAMAMOTO)
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The term "shock" was originally applied to the condition of sudden circulatory break down following a severe injury without gross anatomical damages in the vital organs. As the studies advanced, it was revealed that the etiology and pathology of the condition were much complicated, but now there is the general agreement that "shock" is an unbalanced condition between the circulatory blood volume and the size of vascular bed, as was advocated by the shock subcomittee (1949). Studying various cases of shock in the field of the World War I. COWELL (1918) devided them into primary and secondary type according to the time factor between the cause and the occurrence, and this classification has been generally accepted. However PHEMISTER and LIVINGSTONE (1934) stated that the difference of these two types must be based on etiological factors.

As YAMAMOTO (1952) pointed out, fainting or vasovagal syndrome of LEWIS (1932) occurs reflexively, and hypotension and bradycardia constitute its cardinal symptoms, just as in the case of the primary shock of ARAKI (1949).

In many experimental studies on reflex shock, such as crushing or electrical stimulation of a peripheral nerve trunk, immediate increase of blood pressure was frequently observed (Loven reflex). However PORTER (1908), by application of excessive heat, JANEWAY (1914), by artifical hyperrespiration and manipulation of the intestines and MANN (1914), by prolonged stimulation of posterior root of the 4th lumbar nerve, produced immediate low blood pressure, though it returned to the initial level within a short time.

Since PHEMISTER and SHACHTER (1934) produced hypotension by electrical stimulation of aortic nerve in animals, a great number of studies have been carried out to clarify the influence of the autonomic nervous system on this kind of hypotension. TAKEUCHI (1951) succeeded in producing the reflex shock (hypotension) by injection of tincture of iodine into the pleural cavity or by operative manipulation of carotid sinus of rabbits, and IIDA (1952), by injection of tincture of iodine, hydrochloric acid or acetylcholin into the pleural cavity of cats, and thus they postulated that the reflex shock must be brought about through the vagal nerve.

MINE (1949) observed that when animals were starved for a considerable period of time or inflicted continuously painful stimulations, they fell readily into the state of shock (hypotension). IIDA (1953), in the course of his experiments of pleural shock in cats, demonstrated that freezing of the body caused the predisposing state of shock.

The present study was done to see whether continuous electrical stimulations of a peripheral nerve may predispose the reflex shock, and if so, whether there may be some microscopic change in the spinal cord.

EXPERIMENTAL TECHNIC

Rabbits, about 2 kg in weight, were used. Starved and fixed on a table, sciatic nerve, or brachial plexus or the second branch of trigeminal nerve was exposed and stimulated by means of bipolar electrodes, 1 cm apart from each other, incessantly for 24 hours with square pulses of 2 v 10 c/sec and 20 msec. Subsequently, sciatic nerve or brachial plexus on the contralateral side was crushed violently for 1 minute with a hemostatic clamp, and the following changes of blood pressure and pulse rate were investigated. Blood pressure was recorded graphically by means of a cannula inserted into the femoral artery of the stimulated side. In order to measure pulse rate exactly, electrocardiographs were employed in some cases.

In the course of the experimental studies, every caution had to be payed to prevent hemorrhage and damage to surrounding structures for the purpose of avoiding any additional factor possibly predisposing the shock. Blood pressure and pulse rate were observed for 10 minutes after crushing, and then rabbits were bled to death foh histological examinations.

RESULTS

Series I. Control.

In the animals starved and fixed on a table for 24 hours without any preliminary stimulation, following peripheral nerves were crushed.

A) Crushing of sciatic nerve.

Sciatic nerve was exposed and crushed violently for 1 minute with a hemostatic clamp. (Table 1)

Table 1.

No.		Before crushing	Just after crushing	1 min. after crushing	3 min.	5 min.
1	Blood pressure	87	92	82	78	78
	Pulse rate	308	402	376	360	
2	Blood pressure	76	104	68	76	76
	Pulse rate	280	380	360		
3	Blood pressure	96	136	94	96	66
	Pulse rate	420	480	469	460	460
4	Blood pressure	80	100	80	80	80
	Pulse rate	360		420	410	380
5	Blood pressure	86	112	88	86	88
	Pulse rate	328		416	338	320

B) Crushing of brachial plexus.

Brachial plexus was crushed similarly. (Table 2)

Table 2

No.		Before crushing	Just after cruchhing	1 min. after crushing	3 min.	5 min.
6	Blood pressure	74	110	78	74	74
	Pulse rate	280	312	288	276	294
7	Blood pressure	76	112	86	76	76
	Pulse rate	244		308	288	284
8	Blood pressure	102	126	96	100	100
	Pulse rate	420	438	410	452	438
9	Blood pressure	62	90	66	66	66
	Pulse rate	280		320	428	332
10	Blood pressure	84	100	86	88	90
	Pulse rate	360		430	390	400

In this series, rabbits cried as if they complained of severe pain. Blood pressure increased markedly just after the crushing, but soon returned to the initial level. Pulse rate usually became more frequent, but in some cases changed insignificantly, and in no cases pulse became slower.

Series II. Crushing of a peripheral nerve after the preliminary stimulation of sciatic nerve.

Sciatic nerve was exposed in upper thigh on one side and stimulated preliminarily with square pulses of 2 v 10 c/sec and 20 msec continuously for 24 hours.

A) Crushing of sciatic nerve.

After the preliminary stimulation of sciatic nerve on one side, sciatic nerve on the contralateral side was crushed violently for 1 minute with a hemostatic clamp. (Table 3 and Figs. 1, 2, 3)

Table 3

No.		Before crushing	Just after crushing	1 min. after crushing	3 min.	5 min.
11	Blood pressure	96	78	72	62	74
	Pulse rate	300			264	264
12	Blood pressure	92	64	50	Died 4 min. later.	
	Pulse rate	272		120		
13	Blood pressure	60	68	59	64	60
	Pulse rate	225	260	240	230	
14	Blood pressure	78	94	62	62	62
	Pulse rate	300	290	280	270	

15	Blood pressure	58	84	62	60	60
	Pulse rate	246	320			
16	Blood pressure	78	100	48	20	Died 4 min.
	Pulse rate	240		230		later.
17	Blood pressure	80	104	78	74	
	Pulse rate	318	206	250	250	
18	Blood pressure	92	158	120	78	78
	Pulse rate	270	400		305	290
19	Blood pressure	86	40	75	44	30
	Pulse rate	284	195		320	284
20	Blood pressure	70	96	74	60	68
	Pulse rate	240	326	300	230	270
21	Blood pressure	58	70	64	56	60
	Pulse rate	330	360		360	340

Fig. 1. Animal No. 12.

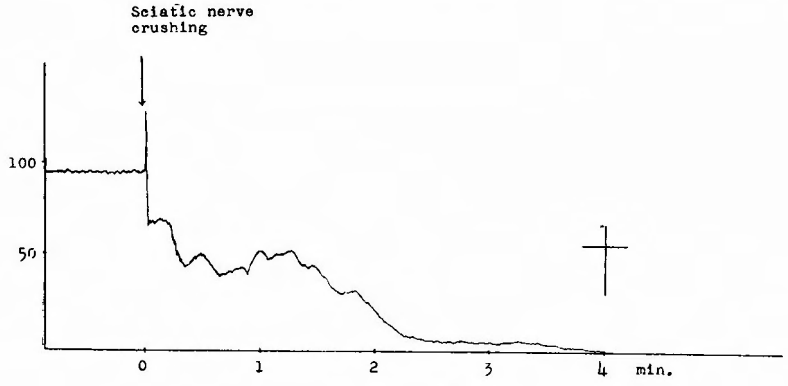
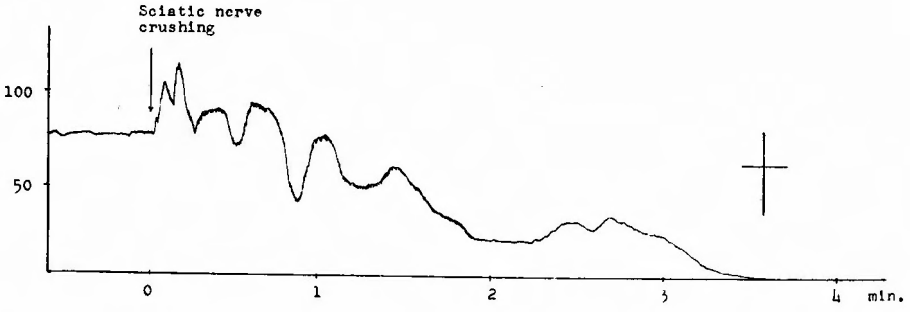
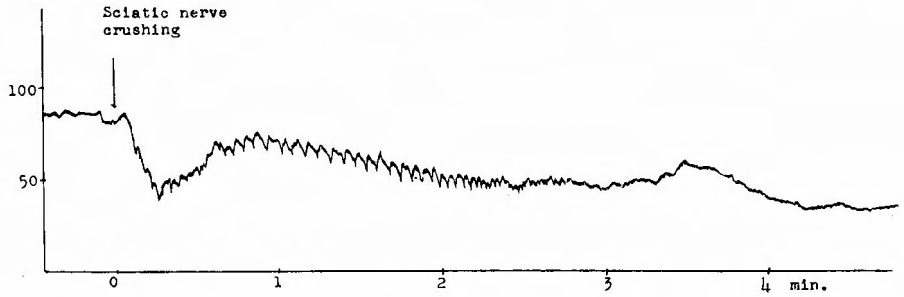


Fig. 2. Animal No. 16.



In 4 (Nos. 11, 12, 16, 19) out of 11 cases, blood pressure fell suddenly and pulse rate became slower immediately after the crushing. No. 11 survived the procedure and the following period of observation, but Nos. 12 and 16 died within

Fig. 3. Animal No. 19.



5 minutes after the crushing and No. 19 was on the verge of death. In the remaining 7 cases, blood pressure increased and in two of them pulse rate decreased.

B) Crushing of brachial plexus.

After the preliminary stimulation of sciatic nerve, right brachial plexus was crushed. (Table 4 and Figs. 4, 5, 6)

Table 4.

No.		Before crushing	Just after crushing	1 min. after crushing	3 min.	5 min.
22	Blood pressure	68	100	50	22	Died 5 min.
	Pulse rate	302	320	220		later.
23	Blood pressure	57	70	43	54	54
	Pulse rate	286	312		310	282
24	Blood pressure	67	54	60	60	60
	Pulse rate	410	370	360	304	
25	Blood pressure	60	90	60	60	60
	Pulse rate	240	312	272	252	236
26	Blood pressure	79	98	76	78	78
	Pulse rate	210	210		210	
27	Blood pressure	70	80	84	72	72
	Pulse rate	210	180		190	190
28	Blood pressure	75	94	54	Died 3 min. later.	
	Pulse rate	262	246			
29	Blood pressure	80	40	58	50	48
	Pulse rate	300	210	180	190	
31	Blood pressure	56	94	54	54	54
	Pulse rate	304	272	286	290	
32	Blood pressure	Died immediately after inserting an electode into the forearm.				
	Pulse rate					

Fig. 4. Animal No. 22.

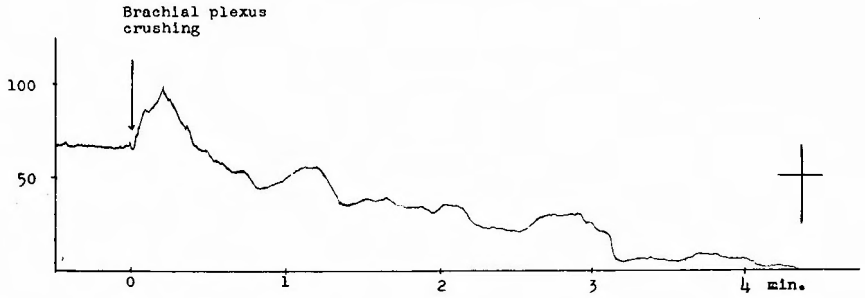


Fig. 5. Animal No. 28.

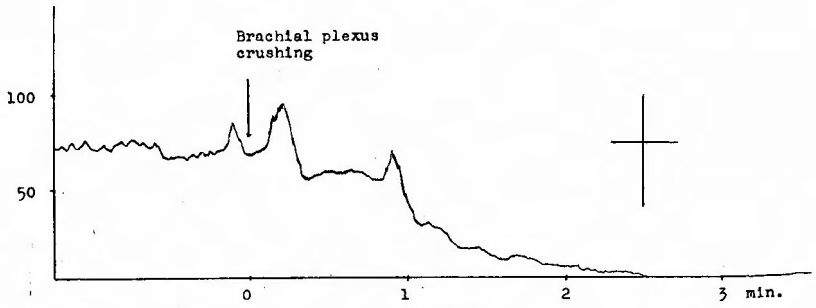
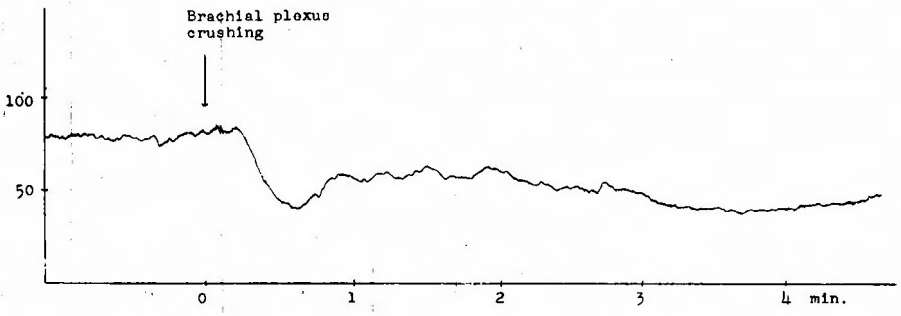


Fig. 6. Animal No. 29.



No. 31 died immediately after the stab of an electrode of the eletrocardiograph into the forleg. It was thought that the animal died of the primary shock, as necropsy revealed no gross damage. In 5 out of the remaining 9 cases (Nos. 22, 23, 24, 28, 29) blood pressure fell markedly. Of these 5 animals two (Nos. 22, 28) died within 5 minutes after the crushing. Though the remaining three survived the procedure, No. 29 was nearly dying when the procedure was over. In all of these five cases except one, which showed slight tachycardia, pulse became slower immediately after the crushing. In other four animals, in which blood pressure increased, pulse rate was accelerated or unchanged.

It has been said that the primary shock does not result in animals from stimulation or crushing of a peripheral nerve. In the present series of experiments too, the primary shock was not caused by the crushing of a peripheral nerve in control animals. But in series II, in which sciatic nerve on one side was stimulated

preliminarily by square pulses, hypotension was observed in 36% on crushing sciatic nerve on the opposite side and in 56% on crushing brachial plexus, and the definite symptoms of the primary shock were observed in 7 cases. Thus it was revealed that the predisposition to the primary shock could be produced by the preliminary continuous electrical stimulation of sciatic nerve on one side.

The next question is that such a predisposition would be produced by the preliminary stimulation of brachial plexus or trigeminal nerve.

Series III. Crushing of a peripheral nerve after the preliminary stmulation of brachial plexus.

In left axilla, brachial plexus was exposed and stimulated continuously for 24 hours with square pulses of 2 v 10c/sec and 20 msec.

A) Crushing of sciatic nerve.

After the preliminary stimulation of brachial plexus, sciatic nerve was crushed. (Table 5 and Figs. 7, 8, 9)

Table 5

No.		Before crushing	Just after crushing	1 min. after crushing	3 min.	5 min.
32	Blood pressure	78	130	74	70	68
	Pulse rate	376	490		386	
33	Blood pressure	72	138	88	60	34
	Pulse rate	290	318		280	
34	Blood pressure	90	104	90	88	88
	Pulse rate	282		314	286	264
35	Blood pressure	103	152	112	104	102
	Pulse rate	310	360		324	310
36	Blood pressure	90	110	98	90	86
	Pulse rate	258	276		266	258
37	Blood pressure	90	146	118	94	90
	Pulse rate	252	308		252	232
38	Blood pressure	98	138	100	95	100
	Pulse rate	272	364		272	256
39	Blood pressure	102	126	102	102	100
	Pulse rate	410	470	430	430	450
40	Blood pressure	94	114	98	92	93
	Pulse rate	456		380	440	432
41	Blood pressure	98	106	100	94	94
	Pulse rate	258	280	290	290	286
42	Blood pressure	114	114	118	116	110
	Pulse rate	290	330		300	286

Fig. 7. Animal No. 32.

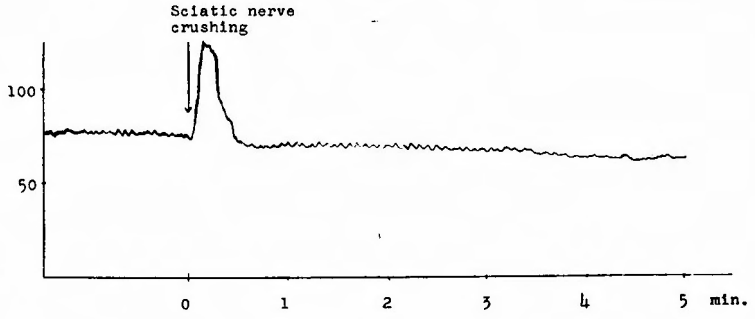


Fig. 8. Animal No. 33.

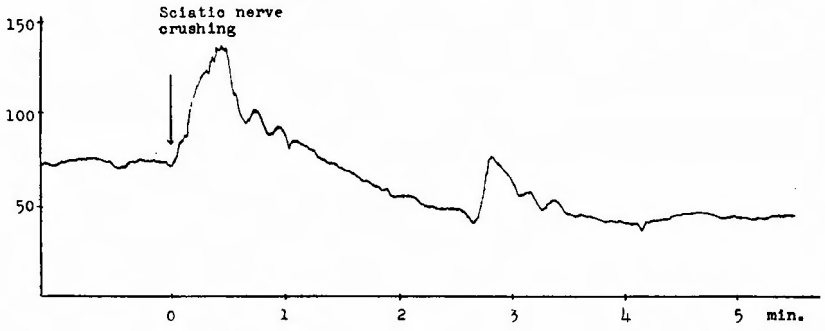
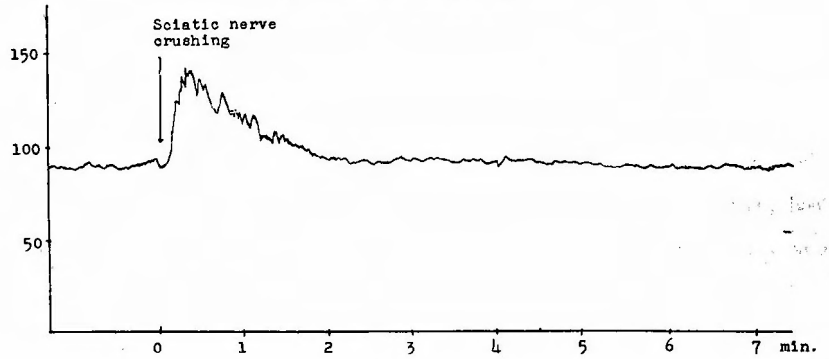


Fig. 9. Animal No. 37.



Sciatic nerve was crushed on the right side in 5 cases and on the left side in 6 cases.

In all cases but one, in which blood pressure decreased 5 minutes after the crushing, blood pressure promptly increased with no remarkable pulse rate change and soon returned to the initial level. All survived the crushing and the following period of observation.

B) Crushing of brachial plexus.

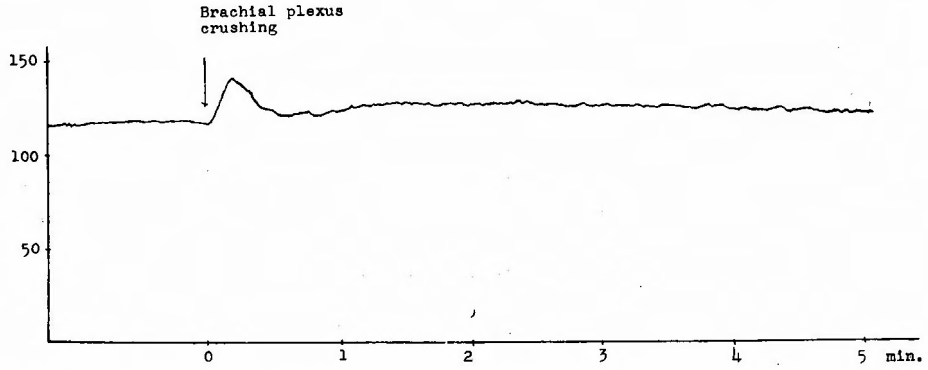
After the preliminary stimulation of brachial plexus on one side, brachial plexus on the contralateral side was crushed. (Table 6 and Fig. 10)

In all cases, blood pressure increased, but in one case the change was not so marked. Pulse rate increased in all.

Table 6

No.		Before crushing	Just after crushing	1 min. after crushing	3 min.	5 min.
43	Blood pressure	111	136	118	118	118
	Pulse rate	270	330		300	286
44	Blood pressure	116	140	124	124	120
	Pulse rate	288		270	270	282
45	Blood pressure	114	116	116	114	114
	Pulse rate	300	306		360	330

Fig. 10. Animal No. 44.



Series IV. Crushing of a peripheral nerve after the preliminary stimulation of the second branch of trigeminal nerve.

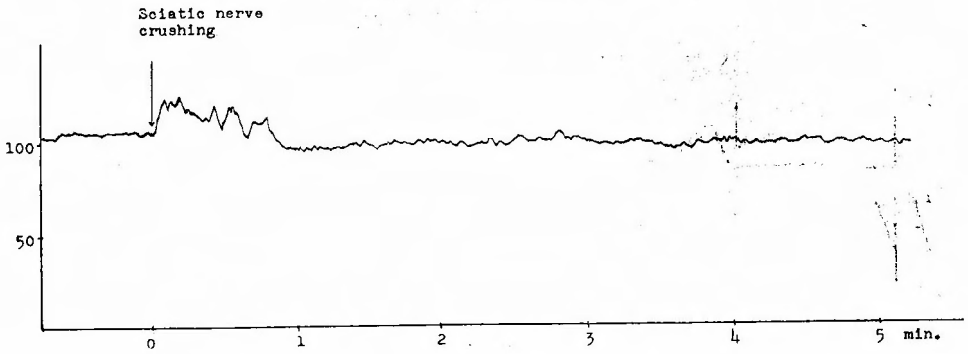
The second branch of trigeminal nerve was exposed and stimulated with square pulses of 2 v 10 c/sec and 20 msec continuously for 24 hours, and then right sciatic nerve was crushed violently for 1 minute by a hemostatic clamp. (Table 7 and Fig. 11)

Table 7

No.		Before crushing	Just after crushing	1 min. after crushing	3 min.	5 min.
46	Blood pressure	96	101	88	95	94
	Pulse rate	252		292	276	256
47	Blood pressure	104	124	96	100	98
	Pulse rate	236	300		236	248
48	Blood pressure	72	84	70	68	68
	Pulse rate	96	136	180	186	180
49	Blood pressure	66	134	108	80	78
	Pulse rate	180	250		230	204

50	Blood pressure	100	120	102	94	95
	Pulse rate	260	360		304	276
51	Blood pressure	98	120	110	104	98
	Pulse rate	260	360		264	246
52	Blood pressure	76	99	88	80	80
	Pulse rate	248	288		272	244
53	Bloodp ressure	81	110	122	84	84
	Pulse rate	230	312	344	296	256

Fig. 11. Animal No. 47.



In all cases, blood pressure rose immediately after the crushing, but soon returned to the initial level, and the shock-like state occurred in no case and pulse rate increased in all.

Comment. When a peripheral nerve was crushed, all animals cried as if they complained of severe pain, and in 6 cases (Nos. 12, 16, 19, 22, 28, 29), blood pressure fell markedly immediately after the crushing. In one case, which died on stab of an electrode of the electrocardiograph, the symptoms of the primary shock, such as cyanosis, midriasis and relation of muscle tone were observed. (Table 8)

Table 8

	Stimulated nerve	Crushed nerve	Shock	Decreased blood pressure	Increased blood pressure	Total	
Series I.	(Control)	Sciatic nerve	0	0	5	5	10
		Brachial Plexus	0	0	5	5	
Series II.	Sciatic nerve	Sciatic nerve	3	1	7	11	21
		Brachial plexus	4	2	4	10	
Series III.	Brachial plexus	Sciatic nerve	0	1	10	11	14
		Brachial plexus	0	0	3	3	
Series IV.	Trigeminal nerve	Sciatic nerve	0	0	8	8	8
		Brachial plexus	—	—	—	—	

HISTOLOGICAL STUDY OF THE SPINAL CORD.

Detailed histological studies of the spinal cords of experimental animals were carried out in 2 cases of series I (Nos. 1 and 9), 4 cases in series II (Nos. 16, 20, 29, 30) and each 2 cases of the series III and IV. (Nos. 37, 38, 46, 48)

As mentioned above, the animals were bled to death for histological examination just after the period of observation and the spinal cords were removed carefully and subjected to dehydration fixation with 99% alcohol. In order to avoid morphological changes of the specimens, which may sometimes occur in the course of fixation, every caution was payed. Then after embedding in celloidin, specimens were cut in 15μ and stained by NISSL's method with thionin solution.

Gross examination of the cords revealed neither hemorrhages nor adhesions.

Series I. Control. Crushing of a peripheral nerve without any preliminary stimulation.

In both cases (Nos. 1 and 9), no pathological findings were present in the cords. (Table 9 and Figs. 12-15)

Series II. Crushing of a peripheral nerve after the preliminary stimulation of sciatic nerve.

In No. 16 which died of shock three and a half minutes after the crushing, NISSL's stain revealed remarkable cellular changes to wide extent above and below the 12th thoracic segment of the cord. In nucl. intermediomedialis and nucl. intermediolateralis, tigrolysis was prominent and widespread almost over the entire cord. However in nucl. ventromedialis, nucl. ventrolateralis, nucl. dorsomedialis and nucl. dorsolateralis in the ventral horn, tigrolysis was present around the level of Th_{12}, and sporadic and less marked. In nucl. dorsalis and nucl. prop. cornu dorsalis in the dorsal horn, nearly no changes were visible. (Table 10 and Figs. 16-22)

In No. 22, which did not fall into the shock state, but showed hypertension and tachycardia immediately after the crushing of sciatic nerve, cellular changes of nucl. intermediomedialis and nucl. intermediolateralis were less marked and sporadic. In motor nuclei of the ventral horn, tigrolysis was scarcely noted. (Table 10 and Figs. 23-27)

In No. 29, which fell into the shock state immediately after the crushing of brachial plexus, tigrolysis was present to a wide extent around the level of L_1, and especially prominent in nucl. intermediomedialis and nucl. intermediolateralis. In the cell groups of the ventral horn, slight tigrolysis was seen at the levels of the lower thoracic segments.

In nuclei of the dorsal horn, nearly no tigrolysis was visible. (Table 10 and Figs. 28-35)

In No. 30, which did not fall into the shock, but showed the temporary hypertension, tigrolysis was present sporadically and less marked. In the cells of the ventral and dorsal horn, the change was scarcely noted. (Table 10 and Figs. 36-39)

Series III. Crushing of a peripheral nerve folllowing the preliminary stimulation of brachial plexus.

In both No. 37 and No. 38 which did not fall into the shock despite the

Table 9

Animal No.			C_1	C_2	C_3	C_4	C_5	C_6	C_7	C_8	Th_1	Th_2	Th_3	Th_4	Th_5	Th_6	Th_7	Th_8	Th_9	Th_{10}	Th_{11}	Th_{12}	L_1	L_2	L_3	L_4	L_5	L_6	L_7	S
1	R	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
9	R	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

crushing of a peripheral nerve after the preliminary stimulation of brachial plexus, blood pressure increased markedly immediately after the crushing and soon returned to the initial level. In No. 37 tigrolysis was sporadic at the levels of C_2, C_6, C_7 and Th_1, and in No. 38 it was present sporadically only in the lower thoracic segments. In both cases, changes were found in nucl. intermediomedialis and nucl. intermediolateralis, but not remarkable. (Table 11 and Figs. 40-45)

Series IV. Crushing of a peripheral nerve following the preliminary stimulation of the second branch of trigeminal nerve.

Both No. 46 and No 47 did not fall into the shock state but showed hypertension and tachycardia despite the crushing of sciatic nerve. In No. 46 slight tigrolysis was found in some cervical segments and in No. 48, in cervical and lumbar segments, however the changes were extremely sporadic. (Table 12 and Figs. 46-47)

Briefly speaking, in shocked rabbits, cellular changes of the cord were more marked and widespread than in not shocked rabbits. HATAGOSHI (1953), in his study on the electrical shock from peripheral nerves, reported of the transneural changes of the cord in rabbits, and in my present study too, tigrolysis of ganglion cells of the cord was noted bilaterally over the wide areas of the cord of rabbits in which sciatic nerve was stimulated preliminarily, while in the cord of rabbits in which brachial plexus instead of sciatic was stimulated preliminarily, changes were bilateral but localized in cervical and upper thoracic segments. In animals in which

Table 10

Animal No.			C_1	C_2	C_3	C_4	C_5	C_6	C_7	C_8	Th_1	Th_2	Th_3	Th_4	Th_5	Th_6	Th_7	Th_8	Th_9	Th_{10}	Th_{11}	Th_{12}	Th_{13}	L_1	L_2	L_3	L_4	L_5	L_6	L_7	S
16	R	ventral horn	−	−	−	÷	+	+	÷	+	−	+	+	+	−	÷	−	−	−	÷	+	+	+	−	÷	+	−	+	÷	−	−
		pars intermedia	÷	÷	+	ǂ	ǂ	+	+	+	+	+	+	+	÷	+	+	+	+	+	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	+	+	+
		dorsal horn	−	−	−	−	÷	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−
	L	ventral horn	−	−	−	−	÷	−	−	÷	−	−	+	÷	−	−	−	−	−	÷	+	+	+	−	+	÷	÷	+	−	−	−
		pars intermedia	÷	÷	÷	+	+	÷	÷	÷	÷	−	+	+	+	+	÷	÷	÷	+	+	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	+	+	+
		dorsal horn	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−
20	R	ventral horn	−	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	+	+	−	−	−	−	−	÷	−	−	−	−	−	−	−	÷	÷	−	−	−	+	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	+	÷	−	−	−	÷	−	+	÷	−	÷	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
29	R	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	÷	−	−	−	÷	÷	+	+	−	−	−
		pars intermedia	−	÷	÷	÷	÷	÷	+	+	+	+	+	÷	+	+	+	+	+	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	ǂ	+	+	+
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	+	÷	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	+	÷	+	+	+	+	÷	+	−	÷	−
		pars intermedia	−	−	÷	÷	−	÷	+	+	÷	÷	+	÷	÷	+	+	+	+	ǂ	ǂ	ǂ	ǂ	ǂ	+	ǂ	ǂ	ǂ	+	+	+
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−
30	R	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	÷	+	÷	÷	−	−	÷	+	+	+	−	÷	−	−	÷	+	−	−	−	−	÷	÷	+	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	÷	−	−	−	÷	−	−	+	+	+	−	÷	−	−	÷	÷	−	÷	−	−	−	÷	+	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

Table 11

Animal No.			C_1	C_2	C_3	C_4	C_5	C_6	C_7	C_8	Th_1	Th_2	Th_3	Th_4	Th_5	Th_6	Th_7	Th_8	Th_9	Th_{10}	Th_{11}	Th_{12}	Th_{13}	L_1	L_2	L_3	L_4	L_5	L_6	L_7	S
37	R	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	+	−	−	÷	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	÷	−	−	+	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
38	R	ventral horn	−	−	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	÷	−	−	−	+	÷	÷	÷	−	+	+	−	÷	−	−	÷	−	−	−	−	−	−	−	÷	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	−	−	−	÷	−	−	−	−	−	−	+	÷	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

Table 12

Animal No.			C_1	C_2	C_3	C_4	C_5	C_6	C_7	C_8	Th_1	Th_2	Th_3	Th_4	Th_5	Th_6	Th_7	Th_8	Th_9	Th_{10}	Th_{11}	Th_{12}	Th_{13}	L_1	L_2	L_3	L_4	L_5	L_6	L_7	S
46	R	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	+	÷	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
		pars intermedia	+	−	−	−	−	÷	÷	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
48	R	ventral horn	−	−	−	−	−	÷	÷	−	−	−	÷	−	−	−	−	−	÷	−	−	−	−	−	+	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	+	+	+	+	÷	−	−	−
		dorsal horn	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	L	ventral horn	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−
		pars intermedia	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	÷	−	−	−	÷	−	−
		dorsal horn	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	÷	−	−	−	−	−	−	−	−	−

trigeminus was stimulated, changes were scarcely visible in the cord.

DISCUSSION

Clinically, it is experienced that, in faintings blood pressure falls alarmingly, pulse rate slows down, respiration becomes slow and shallow, and the patient becomes pale and sometimes unconscious. This condition seems to be analogous to the primary shock However, in animals, TAKEUCHI (1951) showed that the crushing of a peripheral nerve, or testicle or muscles of upper thigh or the stimulation of solar plexus failed to produce the primary shock and PORTER (1908) reported also, that this kind of shock did not result from the burning of skin or the prolonged stimulation of posterior root of a lumbar nerve. In my study too, such a shock did not appear following the crushing of a peripheral nerve in control animals. At least in animals, the primary shock could not be produced by the crushing of a peripheral nerve without the preliminary stimulation.

On the other hand, it is known that many women fell into fainting when frightened by the sound of explosions during the World War II in London.

MINE (1949) and IIDA (1953) demonstrated that animals, exposed to preliminary stress, readily fell into the state of shock. Thus it is supposed that such a predisposing state would be necessary for bringing about the primary shock.

In my study, 7 out of 21 rabbits of the series II, in which sciatic nerve was preliminarily stimulated electrically for 24 hours, fell into the shock state on the crushing of a peripheral nerve. However in animals of the series III and IV, in which brachial plexus or trigeminal nerve was preliminarily stimulated, shock was not caused by the crushing of a peripheral nerve.

In 5 out of 7 shocked rabbits, blood pressure rose markedly just after the crushing. This result is identical with that of Mann, who showed that the vasomoter centre was well functioning even in the shock state.

Microscopically, in the series II, NISSL's stain revealed marked tigrolysis bilaterally in almost entire extent of the cord of the shocked rabbits and the changes were especially remarkable in nucl. intermediomedialis and nucl. intermediolateralis, which are assumed to have the close relationship with autonomic function. But they were sporadic and less marked in not shocked rabbits. In rabbits (not shocked) of the series III and IV, in which brachial plexus or trigeminal nerve was preliminarily stimulated, changes were bilateral but localized in upper thoracic and cervical segments and sporadic and less marked.

These findings show that the degenerative change of the cord produced by the preliminary electrical stimulation of a peripheral nerve should have, if severe, the close relationship with the predisposing state of shock.

Interesting enough in this connection is, "Loven reflex", i. e. the increased blood pressure response to painful stimulation. In cases lacking cellular changes of the cord, increased blood pressure due to vasoconstriction develops on the painful stimulation, whereas decreased blood pressure due to vasodilatation results from the same stimulation in cases with cellular changes of the cord.

SUMMARY AND CONCLUSION

In 53 rabbits, an experimental study of reflex shock was carried out. Seven out of 21 rabbits, in which sciatic nerve was preliminarily stimulated continuously for 24 hours, fell in the state of shock immediately after the crushing of a peripheral nerve. However, it was not the case in other rabbits, in which brachial plexus or trigeminal nerve was preliminarily stimulated.

In the shocked rabbits, NISSL's stain of the spinal cords revealed marked tigrolysis bilaterally in nucl. intermediomedialis and nucl. intermediolateralis of pars intermedia.

From these results, followings are concluded.

1) The primary shock cannot be caused even by the violent stimulation of a peripheral nerve.

2) The long-lasting electrical stimulation preliminarily given to sciatic nerve produces the predisposing state of primary shock.

3) In shocked rabbits, marked tigrolysis is present in nucl. intermediomedialis and nucl. intermediomedialis and nucl. intermediolateralis of the cord to a wide extent from lower thoracic to upper lumbar segments.

4) The predisposing state of shock is caused by the degenerative changes of the cord provocated by the preliminary continuous electrical stimulation.

I wish to thank Prof. Dr. CHISATO ARAKI of the 1st Srugical Division, Kyoto University Medical School, for his guidance and valuable advices.

Illustration of plates

Photomicrographs of spinal cords. (NISSL's stain)

Figs. 12 and **13**. Rabbit, No. 1, Series I. (Control)

Normal cells of nucl. intermediomedialis of the 7th cervical and the 4th thoracic segment.

Figs. 14 and **15**. Rabbit, No. 9, Series I. (Control)

Normal cells of nucl. intermediomedialis of the 12th thoracic segment and of nucl. prop. cornu dorsalis of the 3rd lumbar segment.

Fig. 16. Rabbit, No. 16, Series Ⅱ.

Change in nucl. intermediomedialis of the 12th thoracic segment due to the preliminary electrical stimulation of sciatic nerve. Marked tigrolysis, vacuolisation of the cytoplasm and eccentric location of nucleus are visible.

Figs. 17, 18 and **19**. Rabbit, No. 16, Series Ⅱ.

Change in nucl. intermediomedialis of the 7th cervical, 3rd thoracic and 3rd lumbar segment due to the preliminary electrical stimulation of sciatic nerve. Definite tigrolysis.

Figs. 20, 21 and **22**. Rabbit, No. 16, Series Ⅱ.

Change in nucl. intermediolateralis of the 11th thoracic and the 3rd lumbar segment and nucl. ventromedialis of the 3rd lumbar segment due to the preliminary electrical stimulation of sciatic nerve. Tigrolysis.

Fig. 23. Rabbit, No. 20, Series Ⅱ.

Change in nucl. intermediomedialis of the 2nd lumbar segment due to the preliminary electrical stimulation of sciatic nerve. Tigrolysis.

Figs. 24 and **25**. Rabbit, No. 20, Series Ⅱ.

Cells of nucl. intermediomedialis and nucl. ventrolateralis of the 2nd lumbar segment are unchanged despite of the preliminary stimulation of sciatic nerve.

Figs. 26 and **27**. Rabbit, No. 20, Series Ⅱ.

Cells of nucl. intermediomedialis and nucl. ventromedialis of the 12th thoracic segment are unchanged despite of the preliminary electrical stimulation of sciatic nerve.

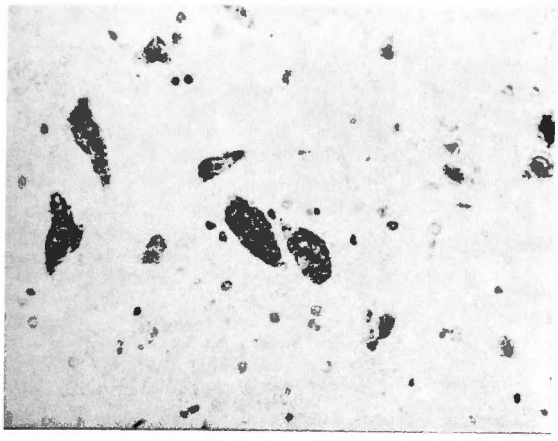


Fig. 12

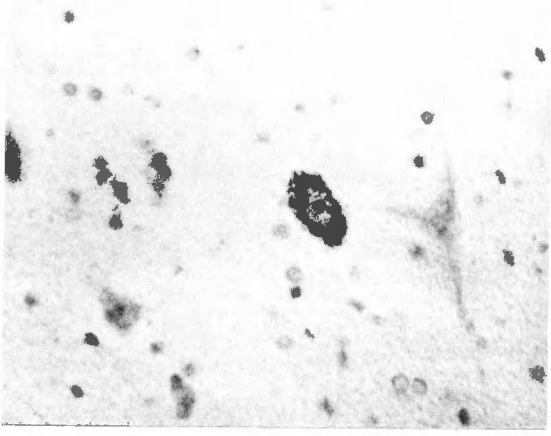


Fig. 13

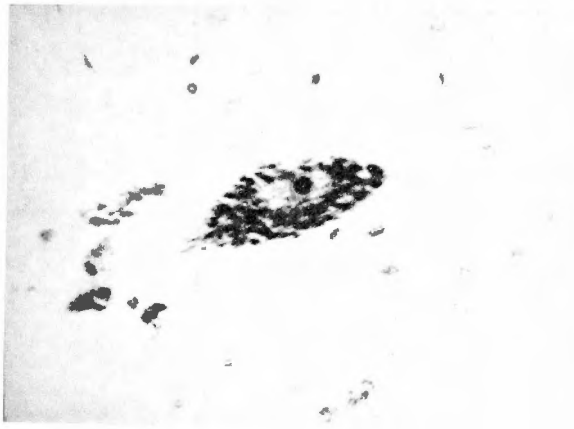


Fig. 14

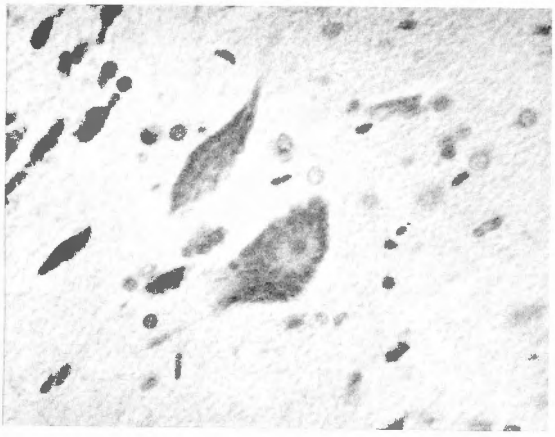


Fig. 15

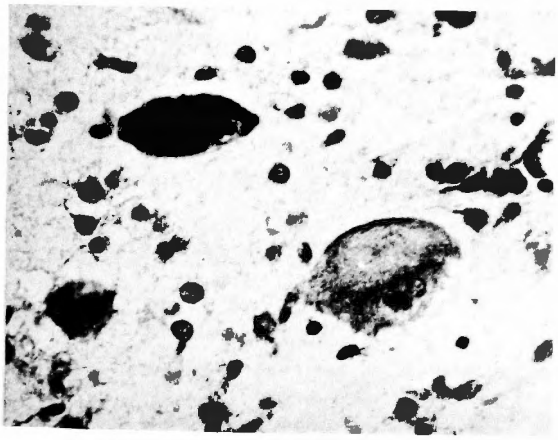


Fig. 16

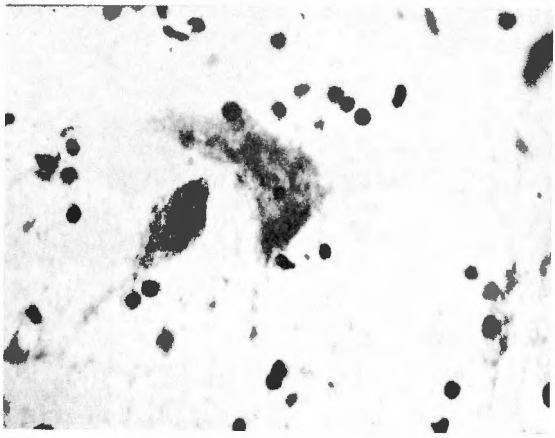


Fig. 17

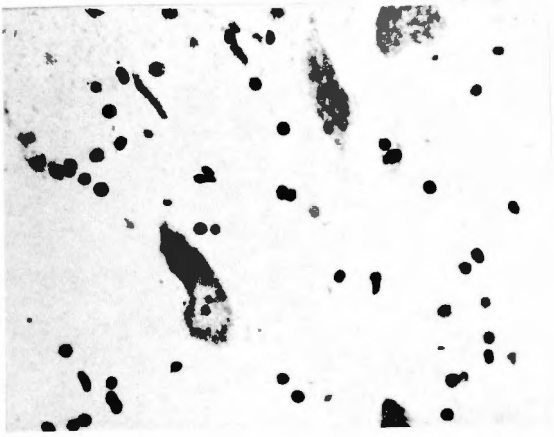


Fig. 18

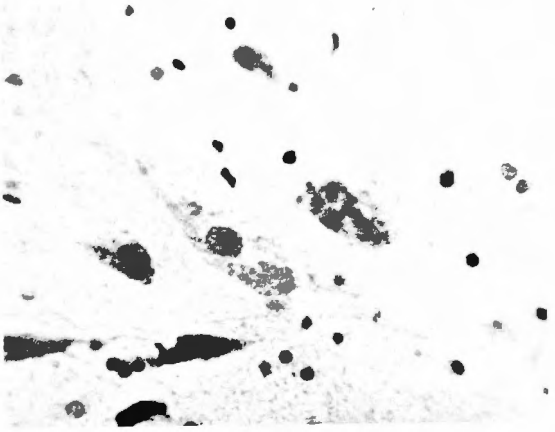


Fig. 19

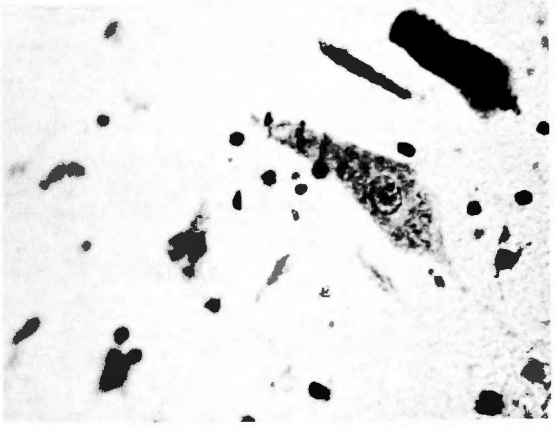


Fig. 20

Fig. 21

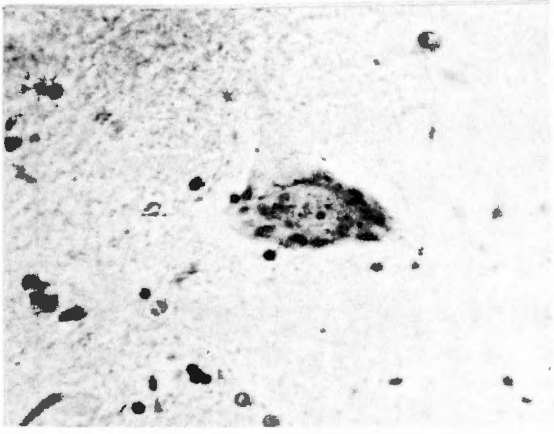


Fig. 22

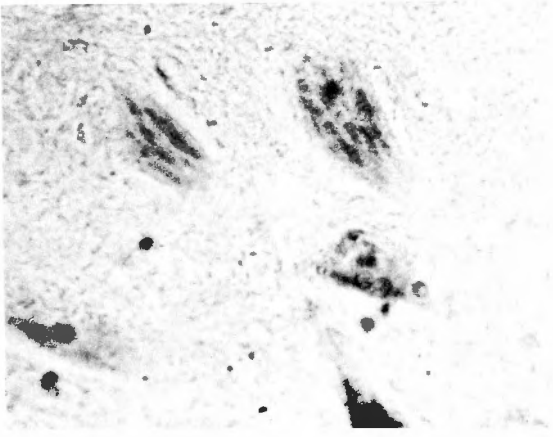


Fig. 23

Fig. 24

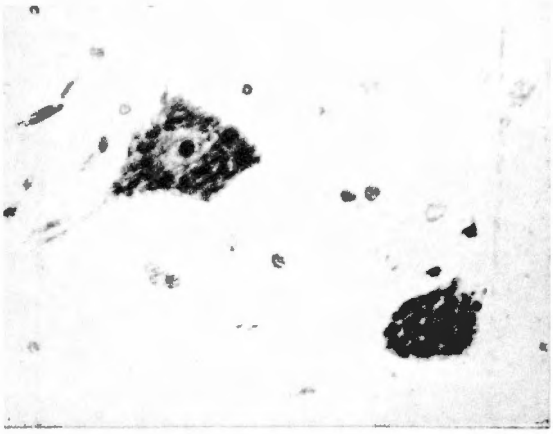


Fig. 25

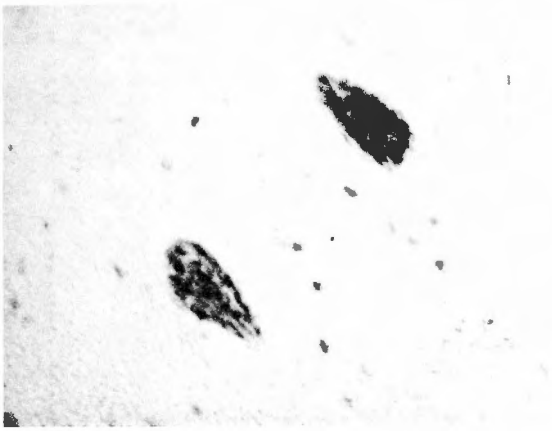


Fig. 26

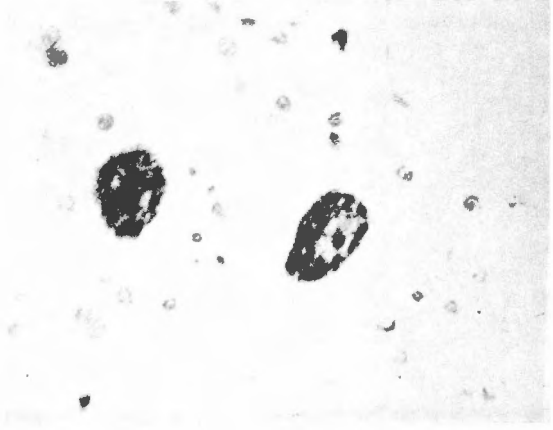


Fig. 27

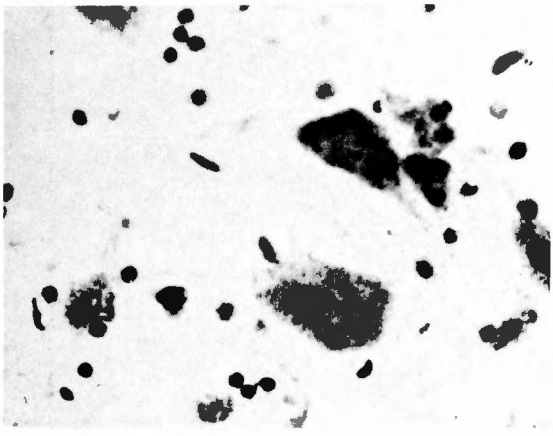


Fig. 28

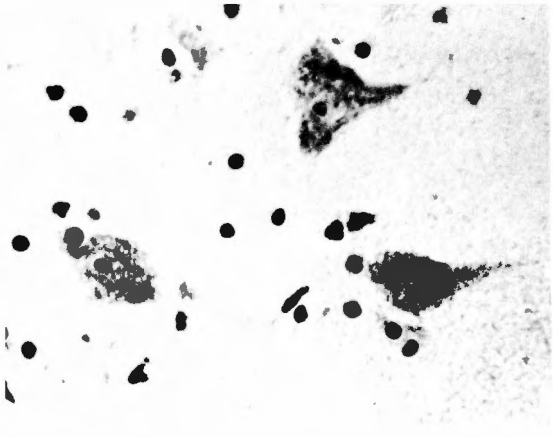


Fig. 29

Fig. 30

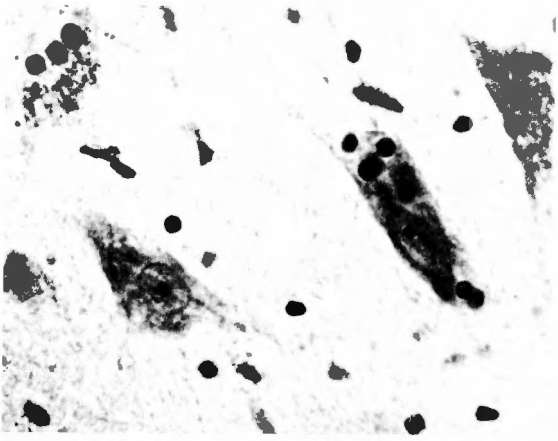


Fig. 31

Fig. 32

Fig. 33

Fig. 34

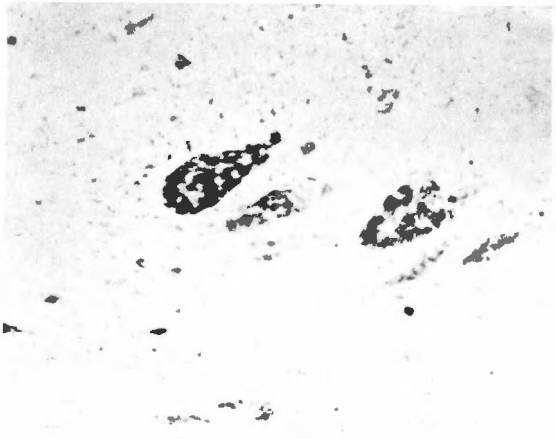


Fig. 35

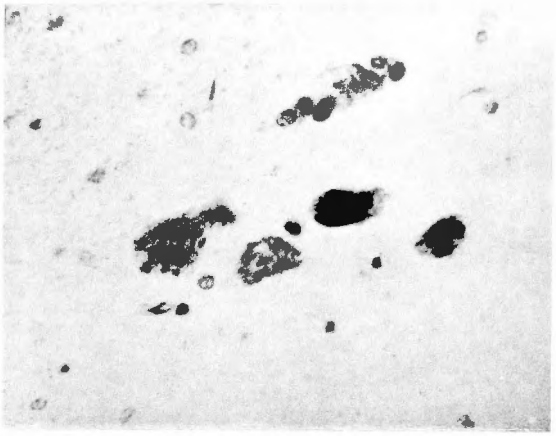


Fig. 36

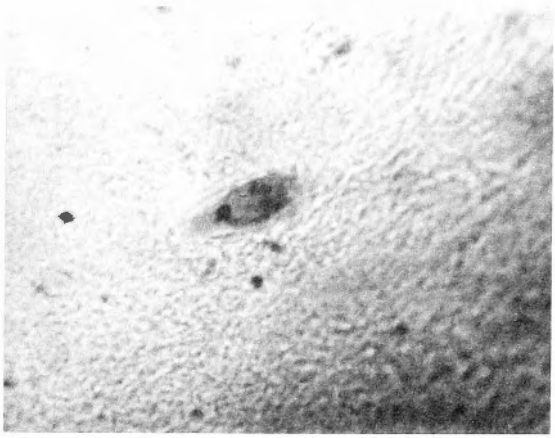


Fig. 37

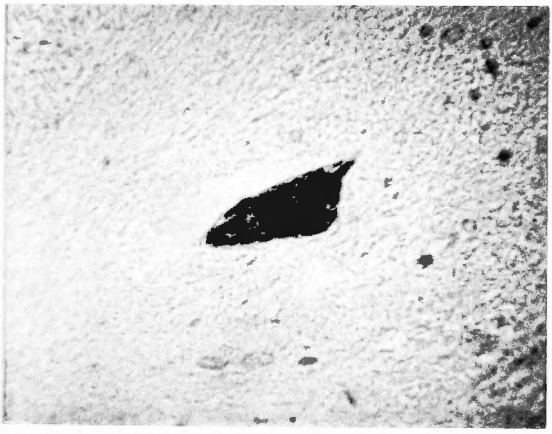


Fig. 38

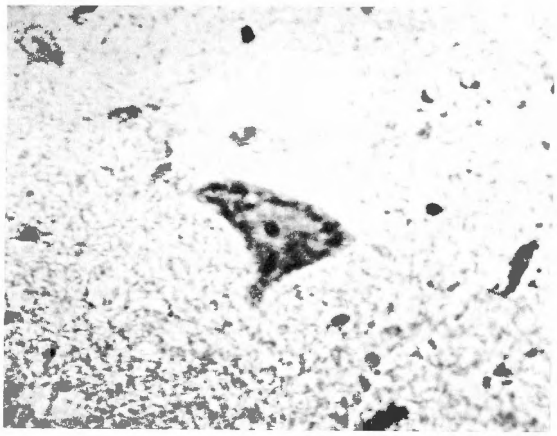


Fig. 39

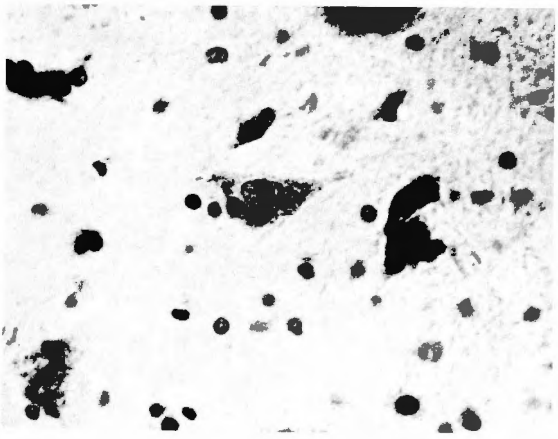


Fig. 40

Fig. 41

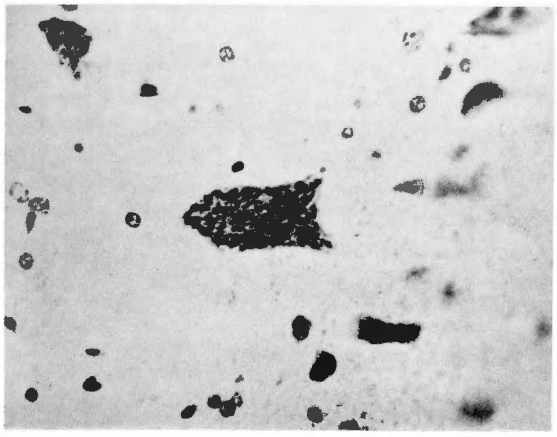


Fig. 42

Fig. 43

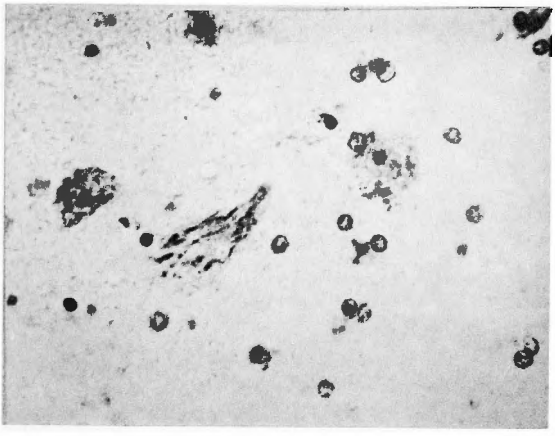


Fig. 44

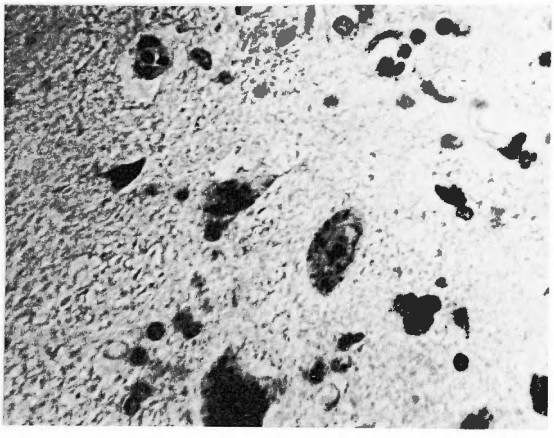


Fig. 45

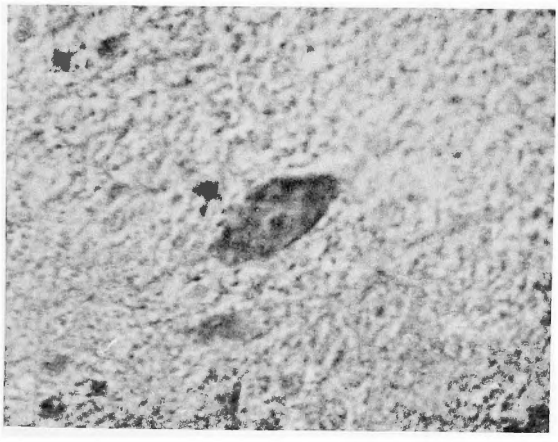


Fig. 46

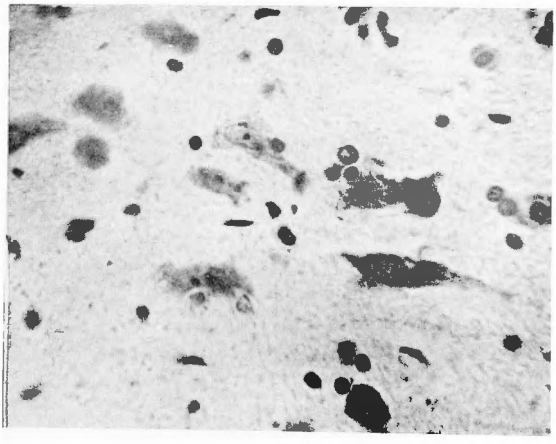


Fig. 47

Figs. 28, 29, 30 and **31**. Rabbit, No 29, Series Ⅱ.

Change in nucl. intermediomedialis of the 6th cervical, the 3rd thoracic, the 13th thoracic and the 3rd lumbar segment due to the preliminary electrical stimulation of sciatic nerve. Tigrolysis.

Fig. 32. Rabbit, No. 19, Series Ⅱ.

Normal cells of nucl. intermediomedialis of the 2nd cervical segment. No change despite of the preliminary electrical stimulation of sciatic nerve.

Figs. 33 and **34**. Rabbit, No. 29, Series Ⅱ.

Normal cells of nucl. ventromedialis of the 6th cervical and the 3rd thoracic segment. No change despite of the preliminary electrical stimulation of sciatic nerve.

Fig. 35. Rabbit, No. 29, Series Ⅱ.

Normal cells of nucl. prop. cornu dorsalis of the 3rd lumbar segment. No change despite of the preliminary electrical stimulation of sciatic nerve.

Fig. 36. Rabbit. No. 30, Series Ⅱ.

Change in nucl. intermediomedialis of the 3rd thoracic segment due to the preliminary electrical stimulation of sciatic nerve. Tigrolysis.

Figs. 37, 38 and **39**. Rabbit, No. 30, Series Ⅱ.

Cells of nucl. intermediomedialis, nucl. dorsalis, nucl. prop. cornu dorsalis of the 10th thoracic segment respectively. In the cells of nucl. intermediomedialis, tigrolysis is seen, but in the cells of nucl. dorsalis and nucl. prop. cornu dorsalis, there is no tigrolysis despite of the preliminary electrical stimulation of sciatic nerve.

Figs. 40 and 41. Rabbit, No. 37, Series Ⅲ.

Cells of nucl. intermediomedialis and nucl. dorsalis of the 6th cervical segment respectively. In the cells of nucl. intermediomedialis, tigrolysis is present, but the cells of nucl. dorsalis are normal.

Figs. 42 and **43**. Rabbit, No. 37, Series Ⅲ.

Normal cells of nucl. dorsalis and nucl. intermediomedialis of the 6th thoracic segment, unchanged despite of the prelimnary electrical stimulation of brachial plexus.

Figs 44 and **45**. Rabbit, No. 38, Series Ⅲ.

Change in nucl. intermediomedialis and nucl. intermediolateralis of the 3rd thoracic segment due to the preliminary electrical stimulation of brachial plexus. Tigrolysis.

Fig. 46. Rabbit. No. 46, Series Ⅳ.

Change in nucl. intermediomedialis of the 6th cervical segment due to the preliminary electrical stimulation of trigeminal nerve. Tigrolysis.

Fig. 47. Rabbit, No. 48, Series Ⅳ.

Change in nucl. intermediolateralis of the 6th cervical segment due to the preliminary electrical stimulation of trigeminal nerve. Tigrolysis.

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和文抄録

反射性ショック及びその準備状態

三重県立大学医学部外科学教室 （指導：山本俊介教授）

武藤浩太郎

家兎を用いて神経反射性ショックの実験を行い次の如き結果を得た．通常動物の末梢知覚神経に強い刺戟を加えてもショックは起らないとされているが，本実験に於ても単に坐骨神経或は上腸神経叢を止血鉗子で挫滅した例では，ショックは起らなかつた．これに反し，予め坐骨神経に24時間持続的電気刺戟を加えておいた21例では末梢神経挫滅により7例にショックを起し得た．上腸神経叢或は三叉神経に予め持続的電気刺戟を加えておいた22例では末梢神経を挫滅してもショックを起し得なかつた．

これ等の家兎の脊髄神経細胞をニッスル氏染色法により検索し，動物の脊髄の中間部細胞に著明な変化を認めた．これ等の実験結果より次の如く結論する．

1) 末梢知覚神経に強い刺戟を加えたのみでは反射性ショックは起らない．

2) 予め坐骨神経に持続的電気刺戟を加えておくとショックが起り易くなる．

3) ショックを起した動物に於ては下胸部より上腰部にかけ広範囲に脊髄神経細胞のニッスル小体が著明に崩壊している．これは Nucl. intermediomedialis 及び Nucl. intermediolateralis に特に著明である．

4) ショック準備状態は持続的疼痛刺戟による脊髄神経細胞の崩壊性変化と密接な関係がある．